

SPINAL CORD INJURY GUIDELINES 2026

Department of Physical Medicine and Rehabilitation/UAMS Brain Injury Program

TELE-REHABILITATION GUIDELINE

Management of Pressure Injuries in Spinal Cord Injury

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I. Definition, assessment, and diagnosis

- A. Definition: Pressure injuries are sores caused by ischemia due to elevated or prolonged pressure to the skin and underlying tissue and occur most often over bony prominences.
1. The pressure injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear forces on the skin.
 2. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.
 3. Pressure injuries can be related by patient's prolonged skin contact with medical devices or other objects.
 4. The recommended term for these injuries is "pressure injury". Note these injuries are also known as pressure ulcers, decubitus ulcers, pressure sores, or bed sores.
 5. Previously, National Pressure Ulcer Advisory Panel (NPUAP) was used to grade pressure ulcer. Now the National Pressure Injury Advisory Panel (NPIAP) is the current standard for pressure injury classification. Notable updates include limiting the term "ulcer" to injuries that includes break in the skin tissue as well as including category for device-related injuries.
 - a. Stage 1 Pressure Injury: Non-blanchable erythema of intact skin:
Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury (DTPI).
 - b. Stage 2 Pressure Injury: Partial-thickness skin loss with exposed dermis: Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel. This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARS), or traumatic wounds (skin tears, burns, abrasions).
 - c. Stage 3 Pressure Injury: Full-thickness skin loss: Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.
 - d. Stage 4 Pressure Injury: Full-thickness skin and tissue loss: Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining

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- and/or tunneling often occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.
- e. Unstageable Pressure Injury: Obscured full-thickness skin and tissue loss: Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (i.e., dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.
 - f. Deep Tissue Pressure Injury (DTPI): Persistent non-blanchable deep red, maroon or purple discoloration: Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury or may be resolved without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.
 - g. Mucosal Membrane Pressure Injury: Mucosal membrane pressure injury is found on mucous membranes with a history of a medical device in use at the location of the injury. These ulcers cannot be staged.¹
- 1. Assessment of individuals with pressure ulcers
 - a. Complete history
 - b. Physical examination and laboratory tests
 - c. Psychological health, behavior, cognitive status, and social and financial resources
 - d. Availability and use of personal care aid
 - e. Positioning, posture and related equipment
 - 2. American College of Surgeons recommends combination of clinical judgement and validated risk assessment tools (Braden scale, Norton Scale, or SCIPUS) to assess pressure injury risks for patients with spinal cord injuries. Although there is no consensus to say which is the best tool.²
 - a. SCIPUS (Spinal Cord Injury Pressure Ulcer Scale), complex multi-item scale, which was developed specifically for pressure injury risk for patients with spinal cord injuries, showed poor predictive validity during inpatient rehabilitation, and likelihood ratios less than 2 indicating it does not substantially improve risk identification beyond chance alone. SCIPUS could only predict pressure ulcers within 2-3 days during acute hospitalization (AUC = 0.85) but did not predict risk over longer periods of time or during rehabilitation.³

¹ Visconti AJ, Sola OI, Raghavan PV. Pressure Injuries: Prevention, Evaluation, and Management. Am Fam Physician. 2023 Aug;108(2):166-174. PMID: 37590857.

² American College of Surgeons Trauma Quality Programs. Best Practices Guidelines: Spine Injury. Chicago, IL: American College of Surgeons; 2022

³ Flett HM, Delparte JJ, Scovil CY, Higgins J, Laramée MT, Burns AS. Determining Pressure Injury Risk on Admission to Inpatient Spinal Cord Injury Rehabilitation: A Comparison of the FIM, Spinal Cord Injury Pressure Ulcer Scale, and Braden Scale. Arch Phys Med Rehabil. 2019 Oct;100(10):1881-1887. doi: 10.1016/j.apmr.2019.04.004. Epub 2019 May 1. PMID: 31054293.

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- b. SCI-PreSORS (SCI Pressure Sore Onset Risk Screening), uses a recursive partitioning model based primarily on Functional Independence Measure (FIM) variables, and outperformed SCIPUS with 98% sensitivity, 75% AUC, and 0.33% false negatives.⁴
 3. A 26-item questionnaire measuring concordance for performing pressure-relief for pressure ulcer prevention in people with Spinal Cord Injury was developed by a SCI group in London, UK.⁵ The Tool proved good reliability and validity. This initial evaluation confirmed the value of the new tool as a novel method for assessing concordance attitude that SCI individuals commonly hold about their pressure-relieving activity to prevent pressure sores.
 4. Assessment of the pressure ulcer
 - a. Anatomical location
 - b. Size (length, width, depth and wound area)
 - c. Stage
 - d. Exudate/odor
 - e. Necrosis
 - f. Undermining
 - g. Sinus tracts
 - h. Infection
 - i. Healing (granulation and epithelialization)
 - j. Wound margins/surrounding tissue
- II. Management and treatment recommendations
 - A. Normal physiological wound healing is a complex, dynamic process which can be divided into four main phases: hemostasis, inflammation, proliferation, and tissue remodeling.^{6, 7, 8}
 1. Immediately following injury, the healing response begins with the activation of the coagulation cascade and the creation of a blood clot. Various chemotactic factors, including platelet-derived growth factor (PDGF), stimulate the migration of blood neutrophils and monocytes, helping to initiate the second phase of healing, the inflammatory response.⁹
 1. Inflammation is responsible for containing, neutralizing, diluting, or walling off the injurious agent or process. Acute inflammation lasts only minutes to days depending on the extent of injury as leukocytes, predominately neutrophils, invade the wound and clear it of debris. As the neutrophils diminish, the wound moves to the chronic inflammation phase with increased activity of macrophages and lymphocytes. This debrides the wound and promotes wound

⁴ Delparte JJ, Flett HM, Scovil CY, Burns AS. Development of the spinal cord injury pressure sore onset risk screening (SCI-PreSORS) instrument: a pressure injury risk decision tree for spinal cord injury rehabilitation. *Spinal Cord*. 2021 Feb;59(2):123-131. doi: 10.1038/s41393-020-0510-y. Epub 2020 Jul 21. PMID: 32694750.

⁵ Liu Lq, Chapman S, Deegan R, Knight SL, Traynor M, Allan HT, Gall A. Development and **preliminary** validation of a tool measuring concordance and belief about performing pressure-relieving activities for pressure ulcer prevention in spinal cord injury. *Journal of Tissue Viability* 30 (2021) 244–249.

⁶ Pressure Ulcers - Chester H. Ho and Kath Bogie DELISA'S PHYSICAL MEDICINE & REHABILITATION: PRINCIPLES AND PRACTICE - 5th Ed. (2010).

⁷ Eming SA, Brachvogel B, Odorisio T, et al. Regulation of angiogenesis: Wound healing as a model. [Proq Histochem Cytochem. 2007;42:115-170.](#)

⁸ Braddock M, Campbell CJ, Zuder D. Current therapies for wound healing: electrical stimulation, biological therapeutics, and the potential for gene therapy. [Int J Dermatol. 1999;38:808-817.](#)

⁹ Moore K. The scientific basis of wound healing. *Adv Tissue Bank*. 2001;5:379-397.

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2. healing. Interleukins and tumor necrosis factor (TNF) concurrently promote angiogenesis and stimulate fibroblasts activity, leading to the wound transitioning to the proliferative phase.¹⁰
 3. Granulation tissue formed during the proliferative phase of healing provides a temporary weak tissue layer in the wound. New blood vessels are produced by budding or sprouting from pre-existing vessels in a process known as neovascularization or angiogenesis. Angiogenesis is essential for granulation tissue formation and improves the overall tissue perfusion (30).
 4. Re-epithelialization: Migration and proliferation of keratinocytes from the wound edges leads to the formation of a thin epithelial layer over the developing granulation tissue is an important healing step in the proliferation phase of wound healing. Wounds left uncovered and open to the air develop a layer of eschar, or scab, over the granulation tissue. Although this layer of dried wound exudates and dead cells protects the wound from environmental contamination, it also prevents keratinocyte migration so that re-epithelialization cannot occur. Re-epithelialization may thus be accelerated by preserving an optimally moist environment.
 5. Collagen repair starts in the proliferative phase and extends into the final remodeling phase. Remodeling of the extracellular matrix (ECM), a combination of extracellular macromolecules and minerals, such as glycoproteins, collagen, enzymes and hydroxyapatite that provides structure and support to surrounding cells, occurs as granulation tissue is reduced via apoptosis.¹¹ Collagen is constantly being synthesized and degraded in order to gain strength through the reorientation of collagen fibers, as wound tissue approaches full strength. Collagen deposition in normal wound healing reaches a maximum by 21 days after the wound is created; however, it may take up to 2 years or more for a scar to completely form. If the remodeling process is disrupted, the equilibrium between ECM deposition and degradation is lost, leading to the development of fibrosis and excessive scarring.¹² Scar tissue contains fewer cells than normal skin and never achieves the same tensile strength as the original skin it has replaced.¹³
- B. Nonsurgical
1. Pressure Off-Loading
 - a. Relief of pressure should be prioritized when first signs of skin breakdown are found. Strategies should include minimizing the time a patient is positioned on the area of skin injury using support surfaces and positioning practices to avoid continued pressure on the ulcer.¹⁴
 - b. Routine repositioning is recommended to avoid injury progression or the development of subsequent pressure injuries, although the ideal frequency is not clearly defined.
 2. Nutritional management
 - a. Adequate nutrition is essential for wound healing. Nutritional optimization should be a priority during initial management.

¹⁰ Anderson JM, Gristina AG, Hanson SR, et al. *Host Reactions to Biomaterials and Their Evaluation*. San Diego, CA: Biomaterials Science Academic Press, Inc.; 1996:165-173.

¹¹ Desmouliere A, Redard M, Darby I, et al. Apoptosis mediates the decrease in cellularity during the transition between granulation tissue and scar. [Am J Pathol. 1995;146\(1\):56-66.](#)

¹² Lorena D, Uchio K, Costa AM, et al. Normal scarring: importance of myofibroblasts. [Wound Repair Regen. 2002;10\(2\):86-92.](#)

¹³ Clark RAF. *The Molecular and Cellular Biology of Wound Repair*. 2nd ed. New York, NY: Plenum Press; 1996.

¹⁴ American College of Surgeons Trauma Quality Programs. Best Practices Guidelines: Spine Injury. Chicago, IL: American College of Surgeons; 2022.

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- b. If patients are malnourished or at risk of malnourishment, the EPUAP/NPIAP/PPPIA guidelines recommend daily dietary intake of 1.25 to 1.5 grams of protein and 30 to 35 kcals per kg of body weight.^{1, 15}
 - c. Adequate hydration should be encouraged for patients when aligned with clinical conditions and patient goals.
 - d. Micronutrient supplementation, such as zinc, arginine and vitamin C may assist in ulcer healing, although the data is inconclusive regarding benefits in patients without existing nutritional deficiency.¹
3. Pain Management
- a. Pain caused from pressure injuries can be constant, severe and can be the most distressing symptom the patient reports. Pain related to pressure injuries can arise from pressure, friction, shear, damaged nerves, inflammation, infection, procedures, excoriations or muscle spasms. Patients with pressure injury should receive ongoing pain assessments, with research showing that pressure injury pain can be assessed using the McGill Pain Questionnaire (MPQ) or select a tool that is relevant to the population (VAS, FACES).¹⁵
 - b. Non-pharmacologic pain management
 - 1) The use of non-pharmacologic strategies when addressing pain related to pressure injuries reflects good practice.
 - 2) Education: Education of the individual, family, and caretakers about the expected duration, as well as a plan on pain management, will enhance understanding and compliance amongst patients and their caretakers, reducing pain overall.
 - 3) Anxiety relief regarding the wound has influences on the physiological and psychological factors related to pain. Anxiety can be relieved through proper communication with the patient, including talking with individuals about their pain, encouraging and answering any questions, allowing active participation and pacing procedures to the individual's preferences.
 - 4) There are non-pharmacologic pain management strategies used in managing pain from pressure injuries, many of these have few studies regarding effectiveness but their benefits in treating acute and chronic pain in both adults and children have been reported. These methods include:
 - i. Positional changes
 - ii. Distraction and conversation
 - iii. Healing touch
 - iv. Music Therapy
 - v. Warmth application
 - vi. Progressive relaxation
 - vii. Meditation
 - viii. Guided imagery
 - ix. Electrotherapy
 - x. VR Immersion
 - c. Pharmacologic pain management
 - 1) Topicals
 - i. Evidence from level 1 studies and lower levels of evidence suggest that the use of a topical opioid can decrease pain by at least four points on

¹⁵ European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. 3rd ed. Washington, DC: National Pressure Injury Advisory Panel; 2019.

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the VAS scoring method. However, there has been insufficient evidence on the use of other topical products that are used to manage pressure related pain, such as NSAIDs or anesthetics.

- ii. Topical opioids generally have minimal systemic effects, itching and irritation have been reported but are equal in frequency to that of placebo therapy.
- iii. Topicals are more useful when treating patients 20 to 30 minutes before a wound procedure.¹⁵

2) Oral medications

- i. Commence pain management with non-opioid medications such as simple analgesics or anti-inflammatory drugs is recommended. These include aspirin, paracetamol, ibuprofen, naproxen, acetaminophen, etc. These drugs act on peripheral nerves to block painful stimuli.¹⁵
- ii. When pain is not addressed with these drugs, a weak opioid agonist may be started in adjunct therapy when initial management is not effective. For severe pain, a stronger opioid may be needed. The long-term use of opioid is associated with adverse outcomes. Evidence suggests that long term opioid use is not effective when managing chronic pressure injury pain.¹⁵
- iii. Adjuvants should be added based on the patient's clinical state and medical diagnoses.
 - 1. These include antibiotics (when infection is causing pain), TCAs or antiepileptics to manage neuropathic pain, and SSRIs to treat depression when increasing pain from the psychological standpoint.¹⁵
- iv. In some cases, a pain specialist may need to be consulted.

4. Cleansing and debridement

- a. Wound cleansing uses fluids to remove surface contaminants and debris, remnants of previous dressings and microorganisms from the wound and peri-wound area, however, does not sterilize the wound.
- b. Use minimal mechanical force when cleansing with gauze.
- c. Use enough irrigation pressure to enhance cleansing without causing trauma to viable tissue. Wound irrigation at a pressure of 4 to 15 psi (pounds per square inch) is recommended.¹⁶
- d. Use normal saline/wound cleansers/potable water.
- e. Avoid long-term use of antiseptics as it delays healing due to cytotoxic properties.
- f. Use hydrotherapy for management of large pressure sores with significant exudate or necrotic tissue with a pulse lavage system. Whirlpool therapy has fallen out of favor due to cross-contamination, size of equipment, and necessity of extensive clean up after each use.
- g. Debride devitalized tissue or eschar from pressure ulcers is a well-accepted practice to decrease bacterial count and promote healing.¹⁷ However, Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed due the risk of exposing bone on the heel and non-healing

¹⁶ Rodeheaver GT, Pettry D, Thacker JG, et al. Wound cleansing by high pressure irrigation. [Surg Gynecol Obstet. 1975;141\(3\):357-362.](#)

¹⁷ Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of current literature. *Ostomy Wound Manage.* 1999;45(suppl 1A):80S-85S; quiz 86S-87S.

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in and ischemic limb. Evaluation for debridement should be considered when fibrinous material, detritus, or debris can no longer be removed gently with fluid and cleansing.

- 1) The initiation of wound treatments such as debridement should occur 20 to 30 minutes after applying topical analgesia to minimize interruptions in patient comfort.
 - 2) Types of debridement include:
 - i. Sharp debridement
 - ii. Autolytic debridement
 - iii. Enzymatic debridement
 - iv. Biological debridement
 - v. Mechanical debridement
5. Dressings
- a. Ensure that ulcer bed is moist and surrounding intact skin is dry to enhance healing.
 - b. Schedule dressing changes to avoid bacterial over-growth.
 - c. Use a dressing that will control exudate but not desiccate ulcer bed or macerate surrounding tissue that would slow healing.
 - d. Loosely fill pressure ulcer cavities with dressing material to avoid dead space; avoid over-packing to ensure good blood flow to healing sore.
 - e. Monitor all dressing placements to ensure they stay in place and stay clean, dry and intact.
 - f. Perform dressing changes on schedule based on individual, ulcer and condition of dressing.
 - g. Consult dressing manufacturer for general information.
 - h. Wound dressing selection
 - i. Basic wound dressings
 - 1) Modern wound dressings have been manufactured and designed to maximize wound healing while promoting comfort of the patient, while also decreasing the need for frequent dressing changes. Studies have shown that healing can take up to 30% longer when compared to modern wound dressings (such as hydrocolloids and foams). Studies have shown there is no significant difference in healing when compared to hydrogels, however the increased need for changes of dressing with gauze is associated with increased costs.¹⁵
 - i. Use moist gauze dressings to support an appropriately moist wound environment when advanced wound dressings are not an available option.
 - 2) Use hydrocolloid, hydrogel and polymeric membrane dressings for non-infected Category/Stage II pressure injuries.¹⁵
 - i. Hydrocolloids examples: DuoDerm, Tegaderm hydrocolloid, Comfeel¹⁸
 - 3) Use hydrogels for non-infected Category/Stage III and IV pressure injuries without significant exudate. Consider use in dry wound beds.¹⁵
 - i. Examples: Curafil, Tegaderm hydrocolloid, Comfeel¹⁸**Error! Bookmark not defined.**
 - 4) Use calcium alginate dressings for Category/Stage II and IV pressure injuries with moderate exudate. Avoid using it in wounds where the wound bed is dry.¹⁵

¹⁸ Rosin NR, Tabibi RS, Trimbath JD, Henzel MK. A primary care provider's guide to prevention and management of pressure injury and skin breakdown in people with spinal cord injury. Spinal Cord Ser Cases. 2020;26(3):177-185. doi:10.46292/sci2603-177.

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- i. Examples: Restore Calcium Alginate, Tegaderm Alginate, Aquacel**Error! Bookmark not defined.**
- 5) Use foam dressings (including hydropolymers) for Category/Stage II and greater pressure injuries with significant exudate. Filler dressings should be used beneath foam dressing in deep ulcers to fill dead space.¹⁵
 - i. Examples: Allevyn, Hydrofera Blue, Hydrocell, Mepilex, PolyMem, Ultra Foam with AFM**Error! Bookmark not defined.**
- 6) Super absorbent dressings can be used in wounds with heavy exudate. Use of these dressings should be cautious, as improper use risks the wound bed drying out.¹⁵
 - i. Examples: ConvaMax, Drawtex, Zetuvit Plus and Biatain Superabsorber**Error! Bookmark not defined.**
- 6. Electrical stimulation combined with standard wound interventions for the treatment of severe (Grade III or IV) pressure ulcers can be helpful if standard care alone is not being successful. The mechanisms by which ES promotes wound healing are not fully understood, leading to a need to optimize delivery of treatment.¹⁹The addition of ES was recommended by the AHCPR (Agency for Health Care Policy and Research) Clinical Practice Guidelines in 1994. Three separate meta-analyses²⁰⁻²² have attempted to consolidate the many varying clinical reports on the success of this technique. A recent study using anodal and cathodal ES administered 50 minutes a day, 5 times a week, showed that ES as a treatment strategy was feasible in a clinical practice to improve peri-wound skin blood flow and promote healing of Stage 2 to Stage 4 pressure sores in patients with neurological injuries.²³ A Canadian review of its benefit noted that it was safe, had low quality evidence of benefit and would increase cost to health care system.²⁴
- 7. Negative pressure wound therapy (NPWT) is based on the theory that the negative pressure facilitates drainage of wound exudates and enhances wound healing through a number of mechanisms.²⁵⁻²⁶

¹⁹ Ennis WJ, Lee C, Gellada K, Corbiere TF, Koh TJ. Advanced Technologies to Improve Wound Healing: Electrical Stimulation, Vibration Therapy, and Ultrasound-What Is the Evidence?

²⁰ Gardner SE, Frantz RA, Schmidt FL. Effect of electrical stimulation on chronic wound healing: a meta-analysis. [Wound Repair Regen. 1999;7\(6\):495-503.](#)

²¹ Cullum N, Nelson EA, Flemming K, et al. Systematic reviews of wound care management: (5) beds; (6) compression; (7) laser therapy, therapeutic ultrasound, electrotherapy and electromagnetic therapy. [Health Technol Assess. 2001;5\(9\):1-221.](#)

²² Akai M, Kawashima N, Kimura T, et al. Electrical stimulation as an adjunct to spinal fusion: a meta-analysis of controlled clinical trials. [Bioelectromagnetics. 2002;23\(7\):496-504.](#)

²³ Polak A, Kucio C, Kloth LC, Paczula M, Hordynska E, Ickowicz T, Blaszcak E, Kucio E, Oleszczyk K, Ficek K, Franek A. A Randomized, Controlled Clinical Study to Assess the Effect of Anodal and Cathodal Electrical Stimulation on Periwound Skin Blood Flow and Pressure Ulcer Size Reduction in Persons with Neurological Injuries. *Ostomy Wound Manage.* 2018 Feb;64(2):10-29.

²⁴ [Lambrinos A](#), [Falk L](#), [Ali A](#), [Holubowich C](#), [Walter M](#). Electrical Stimulation for Pressure Injuries: A Health Technology Assessment. Ontario Health Technology Assessment Series; Vol. 17: No. 14, pp. 1–106, November 2017.

²⁵ Morris GS, Brueilly KE, Hanzelka H. Negative pressure wound therapy achieved by vacuum-assisted closure: evaluating the assumptions. [Ostomy Wound Manage. 2007;53\(1\):52-57.](#)

²⁶ [Orgill DP](#)¹, [Bayer LR](#). Negative pressure wound therapy: past, present and future. [Int Wound J.](#) 2013 Dec;10 Suppl 1:15-9.

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- a. NPWT is proposed to decrease the bacterial load and edema while concurrently promoting improved local circulation and increasing granulation.
- b. NPWT devices consist of a suction pump with foam and occlusive dressing to create negative pressure on the wound being treated. Despite the lack of official guidelines on the use of NPWT, it has been widely used in clinical practice and there have been consensus reports.^{27, 28}
- c. NPWT is indicated when the following clinical criteria are met: Anatomical surfaces that allow a tight seal; Adequately prepared wounds, for example, debrided, free of eschar, and necrotic materials; adequate blood supply to the wound; excessive wound drainage cannot be managed with routine dressings; patient compliance with the device.
- d. Effective NPWT will produce a response within 2 to 4 weeks. Discontinuation is recommended if there is less than 30% wound size reduction after 4 weeks.
- e. Despite its popularity, there has been a struggle to provide scientific evidence to support the use of NPWT for wound healing. A consensus review in 2011 showed evidence base is strongest for the use of NPWT in nonischemic diabetic foot ulcers, followed by pressure sores and weakest in venous leg ulcers.²⁹
8. Therapeutic ultrasound is a deep heating modality that is commonly used for pressure ulcer healing. Its deep heating property is theorized to improve the vascularity of the wound tissues, thus improving healing. However, there is only limited evidence from clinical trials.³⁰
9. Electromagnetic Therapy has been shown to increase the blood flow, collagen formation, and granulocyte infiltration both in vitro and animal models to induce healing, however, clinical trial evidence is again lacking.³
10. Hyperbaric oxygen therapy (HBOT) is not well studied as a treatment option for pressure sores. In a 2015 Cochrane review there was a short term but not a long-term benefit in the treatment of diabetic foot ulcers, but the authors noted various flaws in design and/or reporting that decreased confidence in the results. They felt that more and better designed trials were needed to properly evaluate HBOT in people with chronic wounds. The review article did not find any studies that treated pressure sores with HBOT.³¹ In another review by the British medical journal no studies were found that addressed HBOT and pressure sores.³²
11. Comorbid conditions
 - a. Comorbid conditions can promote wound chronicity and should be considered when creating a comprehensive plan for the patient. Some of these conditions include diabetes, tobacco use, arterial or venous insufficiency.

²⁷ Sibbald RG, Mahoney J; V.A.C. Therapy Canadian Consensus Group. A consensus report on the use of vacuum-assisted closure in chronic, difficult-to-heal wounds. [Ostomy Wound Manage. 2003;49\(11\):52-66.](#)

²⁸ Gupta S. Guidelines for managing pressure ulcers with negative pressure wound therapy. [Adv Skin Wound Care. 2004;17 \(suppl 2\):1-16.](#)

²⁹ S. Vig, C. Dowsett, L. Berg, C. Caravaggi, P. Rome, H. Birke-Sorensen, A. Bruhin, M. Chariker, M. Depoorter, R. Dunn, F. Duteille, F. Ferreira, J.M. Francos Martí nez, G. Grudzien, D. Hudson, S. Ichioka, R. Ingemansson, S. Jeffery, E. Krug, C. Lee, M. Malmsjo, N. Runkel, *, International Expert Panel on Negative Pressure Wound Therapy [NPWT-EP], R. Martin, J. Smith. Evidence-based recommendations for the use of negative pressure wound therapy in chronic wounds: Steps towards an international consensus. *Journal of Tissue Viability* (2011) 20, S1-S18.

³⁰ Baba-Akbari Sari A, Flemming K, Cullum NA, et al. Therapeutic ultrasound for pressure ulcers. [Cochrane Database Syst Rev. 2006;3: CD001275.](#)

³¹ Kranke P, [Bennett MH](#), MartynSt James M, Schnabel A, [Debus, SE](#), Weibel, S. Hyperbaric oxygen therapy for chronic wounds. *Cochrane Database of Systematic Reviews*. 6, 2015.

³² Reddy M. Pressure ulcers: treatment. [BMJ Clin Evid.](#) 2015 Dec 11;2015. pii: 1901.

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- b. Education and treatment regarding tobacco cessation and diabetic management should be assessed during initial evaluation.
 - c. A palliative rather than a curative wound treatment plan may be needed based on the relative comorbid conditions.**Error! Bookmark not defined.**
12. Reassessment
- a. Monitor on a scheduled basis, document changes, and modify treatment if no healing over a 2–4-week period.
 - b. Document status of sore and any changes to pressure ulcer management at least once weekly.
 - c. Review individual risk factors.
 - 1) Impaired mobility is commonly due to paralysis or other concurrent medical conditions, leading to an increased risk of ulcer development, especially in the hospital setting,
 - 2) Nutritional status can be measured by various means: food intake, body weight, body mass index (BMI), serum total protein, albumin, and serum prealbumin. Serum albumin level less than 3.5 mg/dL has also been associated with pressure ulcer development.³³ Serum prealbumin has a shorter half-life than serum albumin, and therefore, is a more sensitive indicator for measuring nutritional status.
 - 3) Anemia has been identified as a risk factor in both the general³⁴ and the spinal cord literature.³⁵ Hemoglobin level below 12.0 to 14.0 g/dL has been associated with the development of pressure ulcers.³⁷
 - 4) The local environment at the skin-support interface, termed the microenvironment, has a localized effect on the risk to pressure ulcer development. This umbrella term covers several factors including temperature, moisture, and acidity.
 - 5) Skin temperature provides an indicator of tissue perfusion. It has been found that immobile patients do not recover preloading skin temperatures after a period of prolonged loading.³⁸ However, elevated body temperatures will raise metabolic activity by increasing the need for cellular oxygen. Animal studies have found that deep tissue damage occurs at 10° above room temperature whereas even higher temperatures caused both cutaneous and subdermal damage.³⁹ In some patient populations, such as those with SCI, dysfunctional

³³ Blaylock B. A study of risk factors in patients placed on specialty beds. [J Wound Ostomy Continence Nurs. 1995;22\(6\):263-266.](#)

³⁴ Bansal C, Scott R, Stewart D, et al. Decubitus ulcers: a review of the literature. [Int J Dermatol. 2005;44\(10\):805-810.](#)

³⁵ Scivoletto G, Fuoco U, Morganti B, et al. Pressure sores and blood and serum dysmetabolism in spinal cord injury patients. [Spinal Cord. 2004;42\(8\):473-476.](#)

³⁶ Fuoco U, Scivoletto G, Pace A, et al. Anaemia and serum protein alteration in patients with pressure ulcers. [Spinal Cord. 1997;35\(1\):58-60.](#)

³⁷ Rochon PA, Beaudet MP, McGlinchey-Berroth R, et al. Risk assessment for pressure ulcers: an adaptation of the National Pressure Ulcer Advisory Panel risk factors to spinal cord injured patients. [J Am Paraplegia Soc. 1993;16\(3\):169-177.](#)

³⁸ Baldwin KM. Transcutaneous oximetry and skin surface temperature as objective measures of pressure ulcer risk. [Adv Skin Wound Care. 2001;14\(1\):26-31.](#)

³⁹ Kokate JY, Leland KJ, Held AM, et al. Temperature-modulated pressure ulcers: a porcine model. [Arch Phys Med Rehabil. 1995;76\(7\):666-673.](#)

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- temperature regulation may produce abnormal variations in micro-environmental temperature.
- 6) Moisture from sweat or incontinence will both super-hydrate the epidermis and alter the acidity. Skin maceration will both soften the stratum corneum and increase the friction coefficient of the epidermis, which promotes adhesion of the skin to the support surface and increases shear, easy sloughing, and ulceration. The net effect is rapid destruction of the epidermis and reduced antibacterial properties.⁴⁰
- d. Evaluate healing process with instrument or other measurement standards.
- C. Common complications of pressure ulcers
1. Non-surgical
 - a. Tissue infection/Osteomyelitis
 - 1) Identify presence of bone tissue in ulcers not responding to treatment. Pay special attention to any protruding bones.
 - 2) Obtain a tissue/bone biopsy for culture if necessary.
 - b. Immobility caused by bedrest and pressure ulcer management. Address:
 - 1) Nutritional deficiencies
 - 2) Dehydration
 - 3) Decreased range of movement (ROM)
 - 4) Deconditioning
 - 5) Spasticity
 - c. Additional skin issues: Clinician must identify and address Hyper-granulation tissue that can impede healing. Clinicians can decrease the amount of granulation with silver nitrate sticks or with sharp debridement techniques at bedside.
 - d. Psychosocial impacts of pressure ulcers and associated immobility. The patient may need the following:
 - 1) Vocational Rehab
 - 2) Therapies: including nutrition counseling, physical and occupational therapies and peer counseling/support groups
 - 3) Psychotherapy/family therapy
 2. Surgical complications
 - a. Infections: Infection is one of the most common surgical complications. Infection can affect the wound acutely or in a delayed fashion. Patients can develop abscess.
 - b. Wound dehiscence and/or wound separation: multiple causes including uncontrolled spasticity.
 - c. Hematoma or seroma: These often develop post-operatively.
 - d. Flap complications or the need for multiple surgical procedures and re-operations. Other flap complications include wound dehiscence, bleeding, hematoma, recurrence, partial necrosis, or total flap necrosis.⁴¹
- D. Surgical referral: Stage I and II pressure ulcers are managed with local wound care. Surgical care is recommended for patients with complex, deep, stage III with undermining tracks or stage IV pressure ulcers that do not respond to standard wound care techniques.⁴¹
1. Preoperative considerations

⁴⁰ Reger SI, Ranganathan VK, Sahgal V. Support surface interface pressure, microenvironment, and the prevalence of pressure ulcers: an analysis of the literature. [Ostomy Wound Manage. 2007;53\(10\):50-58.](#)

⁴¹ Craven BC, Kreder HJ, Noonan VK, et al. Temporal trends in major in-hospital and immobility-related complications following traumatic spinal cord injury: a retrospective cohort study. *Spinal Cord*. 2013;51(5):364-370. doi:10.1038/sc.2012.158

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- a. Debridement: the starting surgical option for most pressure ulcers. During debridement necrotic tissue and bone are removed. Debridement is typically completed with scalpel, electrocautery, rongeur or curette. This surgery often requires anesthesia. It is important to be aware that SCI patients have altered respiratory reserves which may be important for surgical planning. Often, patients are examined for the formation of any potential sinus tracts.²
 - b. Nutritional status: Screen patients for malnutrition using the Nutritional Risk Screening (NRS) or the Spinal Nutrition Screening Tool (SNST). Malnutrition is associated with increased risk of pressure ulcer development, decreased wound healing, and decreased in protection around bony prominences. Nutritional labs to consider include: fasting blood sugar, liver function panel, folate, vitamin B12, vitamin C, omega-3-fatty acids, prealbumin, albumin, total protein, hemoglobin, hematocrit, total lymphocyte count, transferrin, total cholesterol. *Note this is not an exhaustive list of nutrition markers*²
 - c. Bowel regulation: Incontinence is a major contributing factor to risk of pressure ulcers. There are reports that patients with difficult to manage incontinence that contributes to pressure ulcers may be candidates for colostomy.
 - d. Severe spasms/contractures: Management of spasticity can decrease risk of pressure ulcers. Uncontrolled spasticity can cause postoperative complications and disrupt potential flap healing.²
 - e. Comorbid conditions: Spinal cord injury patients develop a variety of organ system dysfunction over time. Important comorbidities to address include cardiovascular, pulmonary, peripheral vascular disease, and endocrine dysfunction, particularly diabetes.²
 - f. Previous ulcer surgery: Complications of previous surgeries must be considered. Some patients with SCI may require flap surgery.²
 - g. Smoking cessation: patients with spinal cord injury develop altered vascular systems. In patients who smoke they have increased risk of post-operative complications and worsening of vascular disease if present.²
 - h. Osteomyelitis management: More common in stage IV pressure ulcers. This condition usually arises due to bacterial contamination of exposed bone. Bone biopsy should be obtained to guide antibiotic treatment. Antibiotic therapy can last for up to 3 months. In patients requiring flap surgery and radical debridement, osteomyelitis should be addressed first.²
 - i. Urinary tract infection (UTI) treatment: Again, incontinence is a major contributing factor in the development of pressure ulcers. Those with difficult to manage incontinence may be candidates for suprapubic catheters or other indwelling catheter placement.
 - j. Heterotopic ossification assessment: can begin as soon as 3 weeks after SCI. This phenomenon occurs below the level of the lesion. The most commonly affected locations include the hip (70-97%) and less commonly the knee. Presenting features of HO include decreased range of motion in affected joints, pain, swelling, erythema, heat, and low-grade fever. In the workup be sure to rule out infection. XR can be obtained. In early phases, triple phase bone scan can be positive. However, this result is not pathognomonic for HO. CT is an additional imaging modality to consider, this modality is useful for pre-surgical planning. HO remains a clinical diagnosis.²
 - k. Anemia management: Chronic spinal cord injury patients can have anemias of various causes. Anemia or chronic disease is common. It is important to obtain a baseline hemoglobin and hematocrit of these patients before surgery is completed.
2. Postoperative care

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- a. Positioning: Optimal positioning is necessary. The highest pressure on the sacrum occurs when patients lie in 90 degrees supine and 90 degrees lateral position. Lowest pressure is in the 30 degrees semi-Fowler and prone positions. Additionally, pressure on the sacrum increases when the HOB is above 30 degrees.²
- b. Air-fluidized bed: Can aid in preventing pressure relief. Standard mattresses have increased risk of development of pressure ulcers. Other Potential pressure relief mattresses include: low-air loss, vascularized bed, pressure relieving heat bed, Clinitron bed, and alternating pressure mattress.²
- c. Sitting protocol after bed rest: There should be a structured return to sitting protocol describing increasing time intervals of sitting. This protocol is often determined by the operating surgeon.
- d. Unloading of surgical area for 4-8 weeks: Immobilization and bed rest is recommended for 2-8 weeks.² However, clinicians must balance the risk of immobilization with the need for patients to engage in safe activity.
- e. Patient education to prevent recurrence of pressure ulcers: Education of patients and caregivers to try to prevent recurrence of pressure ulcers is crucial. An emphasis should be placed on patient re-positioning, proper sitting, pressure relief techniques, wheelchair maintenance and cushion upkeep, daily home monitoring especially of high-pressure areas, transfer quality.² Patients may require intensive outpatient therapy or even inpatient rehabilitation to incorporate the above strategies.

III. Risk factors, prevalence, prevention and nutrition

- A. Risk Factors: Risk factors for development of pressure ulcers in SCI is multifactorial and includes: underweight BMI, history of smoking, decreased activity levels, incontinence, pulmonary disease, decreased albumin, decreased mobility, prolonged paralysis, increasing age, use of medications for sleep, impaired cognitive function, diabetes, living in a hospital or nursing home, spasticity, renal disease, and low hematocrit.⁴² It is important to note that the above list is not exhaustive and that over 200 risk factors have been identified.⁴³ In fact, one approach to simplify the most common risk factors of pressure ulcers development: is “the big 3” incontinence, immobility, and altered skin integrity/skin sensation.²
 1. Protective factors include: completing a college degree, being married, being employed, exercising, and a healthy diet.
 2. To aid in identification of those at risk for pressure ulcers: Document risk in admission by using approved assessment scales. Additionally, reassess on routine basis. The most important emphasis is on identifying high-risk populations and engaging in prevention.
- B. Prevalence:
 1. In the US, pressure ulcers affect 13-17% of the overall population. The prevalence of pressure ulcers increases in the SCI population. Pressure ulcers are costly for the healthcare system and cost over 3 billion dollars per year annually.⁴³
 2. In a multi-centered cohort study by the SCI model systems⁴⁴, they followed 3361 people with traumatic SCI injured between 1986 and 1995 on a yearly basis through 2002. Prevalence in the first 10 years ranged from 11.5% in year one to 14.1% in year ten, but by year 15 the

⁴² Mortenson WB, Miller WC; SCIRE Research Team. A review of scales for assessing the risk of developing a pressure ulcer in individuals with SCI. *Spinal Cord*. 2008;46(3):168-175. doi:10.1038/sj.sc.3102129

⁴³ Groah SL, Schladen M, Pineda CG, Hsieh CHJ. Prevention of pressure ulcers among people with spinal cord injury: a systematic review. *PM R*. 2015;7(6):613-636. doi:10.1016/j.pmrj.2014.12.007

⁴⁴ Chen Y, DeVivo MJ, Jackson AB. Pressure Ulcer Prevalence in People With Spinal Cord Injury: Age-Period-Duration Effects. *Arch Phys Med Rehabil* 2005;86:1208-13.

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prevalence was 21.0%. A different study showed that during the initial acute and rehabilitation hospitalization after SCI 27-40% of people will develop a pressure ulcer.

3. In summary, the risk of pressure ulcers appears to be steady during the first 10 years and increases to 15 years post-injury. Pressure ulcers affect marginalized populations at a higher rate including: the elderly, African Americans, those with less than secondary education, the unemployed, and patients with complete spinal cord injury. Pressure ulcers are also common in single people who are not married, those with a previous history of pressure ulcers, re-hospitalization, prior nursing home stays, and multiple medical co-morbidities.
- C. Prevention: Prevention of pressure ulcers in the spinal cord population requires a multidisciplinary team approach. Additionally, an important part of pressure ulcer prevention is identifying those at high-risk and continuing to monitor for potential progression of the wound. Spinal cord injury causes and spinal cord injury level have no significant effect on the risk of pressure ulcer development. Below are multiple prevention strategies for pressure ulcer prevention in the spinal cord injury population:
 1. Avoid prolonged immobilization when possible.
 2. Ensure adequate pressure relief.
 3. Use intraoperative pressure reduction strategies.
 4. Avoid improper seating position in wheelchair or use of improper wheelchair cushion.
 5. Conduct daily visual and tactile inspections of bony prominences with increased risk of pressure ulcer development: at the ischium, sacrum/coccyx, trochanters and heels.
 6. Ensure proper wheelchair maintenance. Research has shown that wheelchair breakdowns in wheelchair users with SCI can lead to injury, being stranded, or an inability to attend medical appointments, school, or work. Additionally, breakdowns are associated with increased chances of re-hospitalization and pressure injury development, worsening pain and a decrease in self-perceived health.⁴⁵
 7. Positioning: Turning and repositioning in bed every 2 hours is the standard of care. Additionally, avoid the stretching/folding of soft tissues aiming to prevent shearing skin with reposition. Avoid side-lying at 90 degrees due to increased pressure on the trochanteric bursa. 30-degree side-lying on the trochanter is preferred. Recent studies showed a 4-hour turning schedule combined with use of a pressure reducing mattress was associated with the lowest risk of pressure ulcer occurrence across groups.⁴³ However, controlling for confounding variables did not allow a specific conclusion based on mattress type and turn ratio. In summary, prevention of pressure ulcers remains a clinical challenge. Repositioning frequency should be individualized based on clinical judgment, patient bed type, activity and mobility levels.
 8. Environmental support: pressure reducing support surfaces, prevent moisture accumulation on or around the skin, avoid temperature elevation of the skin, use pillows or cushions for unloading bony prominences (do not use donut-type devices as they will surround the sore and cut off blood supply to the area in the middle of the donut); use supportive surfaces for the bed or the wheelchair to improve positioning.
 9. Seating: An appropriate wheelchair and an effective cushion are essential to provide a seating system that will minimize the risk for pressure ulcer development. There is a wide and continuously increasing range of pressure relief cushions available. The main classes of pressure relief cushions are foam, viscoelastic gel, or flotation with air. Some cushions will combine two or more material classes to achieve improved pressure relief properties, in combination with postural stability. USA TechGuide is an online resource maintained by the

⁴⁵ Hogaboom NS, Worobey LA, Houlihan BV, Heinemann AW, Boninger ML. Wheelchair breakdowns are associated with Pain, Pressure Injuries, Rehospitalization, and Self-Perceived Health in Full-Time Wheelchair Users With Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation 2018;99:1949-56.

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United Spinal Association that provides complete and constantly updated reviews of currently available pressure relief cushions.⁴⁶ Again, Donut-rings should not be used as pressure relief cushions because, although pressure is relieved at the center of the ring, pressure distribution around the perimeter leads to high regional pressures and blood flow occlusion, thus slowing the healing process.

10. **Mattress Types:** Static support mattresses are appropriate for patients without a pressure ulcer. Static mattresses also can be used in patients with pressure ulcers who can be positioned so that no load is applied to pressure ulcers at any time. A different option is dynamic mattresses. Dynamic mattresses include low air loss mattresses and air-fluidized beds and are frequently only used for patients with existing pressure ulcers, due primarily to high usage costs. Regardless of mattress type, immobile patients are at an increased risk of pressure ulcer development. The recommended 2-hourly turning schedule for pressure relief sometimes poses a staffing challenge. If loading over an existing pressure ulcer cannot be avoided at all times, there are multiple stage 3 pressure ulcers or there is a large stage 4 sore, then a dynamic support mattress should be utilized.
11. **Pressure relief:** Use an individualized pressure relief technique and a pressure-reduced seating system. Conduct pressure relief technique every 15 -30 minutes when sitting in a wheelchair. This can be done using the arms for a side or forward leaning technique or if unable a manual versus power weight shift system can be used with either a tilt or recline system or an alternating pressure cushion.
12. **Gluteal Neuromuscular Electrical Stimulation System** can regularly stimulate muscles to increase the health of the muscle and surrounding soft tissues. Bogie et al. have investigated the use of a 4-channel gluteal electrical stimulation system (GSTIM), specifically designed to decrease risk factors associated with pressure ulcer development for individuals with SCI.⁴⁷ Subjects with a GSTIM system showed statistically significant decreases in ischial region pressure over time, together with increased gluteal cross-sectional area and enhanced regional blood flow. Sacral nerve root stimulation or surface FES can induce sufficient gluteus maximus contraction and significantly reduce ischial pressure.⁴⁸
13. **Activity:** Minimizing bedrest for pressure ulcer treatment is recommended as prolonged bedrest can lead to many complications including risk of blood clots of various types including deep venous thromboembolism and pulmonary embolism. Thus, effective clinical management without bedrest for patients with pressure ulcers is desirable. Prone trolleys allow patients to gain some mobility but provide only limited function and mobility in the community environment. Standing wheelchairs or standing frames can provide full, extended pressure relief over pelvic region wounds, thus allowing remobilization of individuals with existing pressure ulcers. Standing wheelchairs also have multiple systemic health benefits including increases in lower extremity range of motion, upper extremity strength, bladder, and other systemic functions together with decreased spasm and pain.⁴⁹ Dynamic wheelchairs and cushions provide an alternative approach to a full standing wheelchair. A dynamic

⁴⁶ USA Tech Guide. Cushion reviews. Accessed April 8, 2018. http://www.usatechguide.org/cushion_reviews.php

⁴⁷ Bogie KM, Reger SI, Levine SP. Therapeutic applications of electrical stimulation; wound healing and pressure sore prevention. *Assist Technol.* 2000;12:50-66.

⁴⁸ Liu LQ, Ferguson-Pell M. Pressure Changes Under the Ischial Tuberosities During Gluteal Neuromuscular Stimulation in Spinal Cord Injury: A Comparison of Sacral Nerve Root Stimulation With Surface Functional Electrical Stimulation. *Arch Physical Medicine and Rehabilitation* 2015;96:620-6.

⁴⁹ Dunn RB, Walter JS, Lucero Y, et al. Follow-up assessment of standing mobility device users. *Assist Technol.* 1998;10(2):84-93.

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- configuration wheelchair varies individual system components in order to relieve pressure over areas of tissue breakdown.⁵⁰
14. Dynamic wheelchairs and cushions: provide an alternative approach to a full standing wheelchair. A dynamic configuration wheelchair varies individual system components to relieve pressure over areas of tissue breakdown.⁴⁹ Dynamic wheelchair cushions provide a means to provide automatic weight-shifting due to cyclic inflation and deflation of air cells in high-load regions and may provide benefits similar to those achieved using an advanced wheelchair system.
 15. Additional protective factors: In a qualitative study of medically underserved individuals, eight types of potentially protective factors were identified: meaningful activity, motivation to prevent negative health outcomes, stability/resources, equipment, communication and self-advocacy skills, personal traits, physical factors, and behaviors/activities.⁵¹
- D. Nutrition should be maximized through dietary intake or if needed supplemental feeding and supplements. The effect of nutrition can be monitored by the following techniques:
1. Anthropometric indicators: Key measurements reported in studies include weight, height, BMI, mid-upper arm circumference (MUAC), calf circumference, and skinfold thickness, used to evaluate nutritional status in patients with pressure ulcers.
 - a. Unintentional weight loss is consistently identified as a major phenotypic indicator of malnutrition and an important predictor of pressure injury development.
 - b. BMI and pressure ulcer risk: Evidence indicates a U-shaped relationship between BMI and pressure injury risk, with underweight individuals demonstrating the highest risk, while overweight status may show a protective effect.⁵²
 - c. malnutrition substantially increases risk, with studies reporting up to 11-fold higher odds of pressure injury development.
 - d. Muscle mass assessment: Calf circumference and mid-upper arm circumference are frequently reported as practical indicators of muscle mass, with high sensitivity for malnutrition detection, particularly in settings without body composition devices.
 - e. Additional measures: Skinfold thickness may be used to estimate fat stores, while handgrip strength provides supportive information on muscle function and nutritional status.⁵³
 2. Laboratory monitoring: Laboratory tests are used to assess nutritional status, inflammatory activity, and micronutrient deficiencies in patients with pressure injuries. Recommended laboratory tests include:
 - a. Hemoglobin
 - b. Vitamin D
 - c. Zinc

⁵⁰ Makhsous M, Rowles DM, Rymer WZ, et al. Periodically relieving ischial sitting load to decrease the risk of pressure ulcers. *Arch Phys Med Rehabil.* 2007;88(7):862-870.

⁵¹ Sleight AG, Cogan AM, Hill VA, Pyatak EA, Díaz J, Floríndez LI, Blanchard J, Vigen C, Garber SL, and Clark FA. Factors protecting against pressure Injuries in medically underserved adults with Spinal Cord Injury: A Qualitative Study. *Top Spinal Cord Inj Rehabil* 2019;25(1):31– 40.

⁵² Alipoor E, Mehrdadi P, Yaseri M, Hosseinzadeh-Attar MJ. Association of overweight and obesity with the prevalence and incidence of pressure ulcers: a systematic review and meta-analysis. *Clin Nutr.* 2021;40(9):5089-5098. doi:10.1016/j.clnu.2021.08.006

⁵³ Schuetz P, Seres D, Lobo DN, Gomes F, Kaegi-Braun N, Stanga Z. Management of disease-related malnutrition for patients being treated in hospital. *Lancet.* 2021;398(10314):1927-1938. doi:10.1016/S0140-6736(21)01451-3

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- d. Folate
 - e. Vitamin B12
 - f. Vitamin C
 - g. Ferritin
 - h. CRP
 - i. Note that protein markers such as albumin and prealbumin are negative acute phase reactants that decrease during inflammation rather than reflecting malnutrition. Therefore, it should not be used as direct indicators of nutritional status.⁵⁴
3. Adequate nutritional intake to meet needs:
- a. Calories: 30 to 35 kcal per kg of body weight daily for those who are malnourished or at risk.
 - b. Protein: recommend 1.25 to 1.5 g of protein per kg of body weight
 - c. Micronutrients: (include vitamins A, C, D, E, K, and B complex, along with trace elements such as zinc, selenium, copper, iron, and manganese.)⁵⁵
 - d. Diet: heart-healthy nutrition plan focusing on fruits, vegetables, whole grains, low-fat dairy, poultry, fish, legumes, and limiting saturated fats to less than 6% of total calories.
 - e. Adequate hydration

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers

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⁵⁴ Evans, D. C., Corkins, M. R., Malone, A., Miller, S., Mogensen, K. M., Guenter, P., & Jensen, G. L. (2021). The use of visceral proteins as nutrition markers: An ASPEN position paper. *Nutrition in Clinical Practice*, 36(1), 22–28. <https://doi.org/10.1002/ncp.10588>

⁵⁵ Kassym, L., Zhetmekova, Z., Kussainova, A., Semenova, Y., Vetrova, A., Nurzhan, S., Sarbassova, G., Akhmetova, A., Orazalina, A., Uzbekova, S., & Bjørklund, G. (2025). Pressure ulcers and nutrients: From established evidence to gaps in knowledge. *Current Medicinal Chemistry*, 32(27), 5630–5657. <https://doi.org/10.2174/0109298673322825241018174928>